

UC Davis

UC Davis Previously Published Works

Title

Bacterial Pneumonia in Dogs and Cats: An Update.

Permalink

<https://escholarship.org/uc/item/5b61k368>

Journal

The Veterinary clinics of North America. Small animal practice, 50(2)

ISSN

0195-5616

Author

Dear, Jonathan D

Publication Date

2020-03-01

DOI

10.1016/j.cvsm.2019.10.007

Copyright Information

This work is made available under the terms of a Creative Commons Attribution-NonCommercial-NoDerivatives License, available at <https://creativecommons.org/licenses/by-nc-nd/4.0/>

Peer reviewed

ARTICLE TITLE

Bacterial Pneumonia in Dogs and Cats

AUTHOR NAMES AND DEGREES

Jonathan D. Dear, MAS, DVM
Diplomate, American College of Veterinary Internal Medicine (Small Animal Internal Medicine)

AUTHOR AFFILIATIONS

Assistant Professor of Clinical Internal Medicine
Department of Medicine and Epidemiology
University of California, Davis
Davis, California

AUTHOR CONTACT INFORMATION

University of California
One Shields Avenue
Davis, CA 95616
jddear@ucdavis.edu
@jddear

CORRESPONDING AUTHOR

Jonathan D. Dear

DISCLOSURE STATEMENT

The Author has nothing to disclose.

KEY WORDS

Bacterial pneumonia, lower respiratory tract infection, canine, feline, lower airway disease

KEY POINTS

- Clinically, bacterial pneumonia is diagnosed much more commonly in dogs than in cats, though it is likely underrecognized in cats.
- Viral infection followed by bacterial invasion is common in young dogs while aspiration pneumonia and foreign body pneumonia appear to be more common in older dogs.
- Clinical signs can be acute or chronic and do not always reflect the underlying respiratory condition.
- Definitive diagnosis requires detection of intracellular bacteria in airway cytology or clinically significant bacterial growth from an airway sample, although relevant clinical findings can also support the diagnosis.

- Treatment requires identification of underlying and management of diseases associated with pneumonia, appropriate antimicrobial therapy, and control of airway secretions.

SYNOPSIS

Bacterial pneumonia is a relatively common clinical diagnosis in dogs but seems to occur less often in cats. Underlying etiologies include viral infection, aspiration injury, foreign body inhalation and defects in clearance of respiratory secretions. Identification of the specific organisms involved in disease, appropriate use of antibiotics and adjunct therapy, and control of risk factors for pneumonia improve management.

Outline

Introduction

Classification of pneumonia

- Aspiration

- Canine infectious pneumonia

- Foreign body

- Nosocomial

- Immune dysfunction

Clinical signs

Physical examination

Diagnosis

- Hematology

- Pulmonary function testing

- Thoracic radiography

- Advanced imaging

- Bronchoscopic evaluation

- Bronchoalveolar lavage

- Microbiology

Treatment

Prognosis

Introduction

Bacterial pneumonia remains one of the most common clinical diagnoses in dogs with acute or chronic respiratory disease. Research suggests a complex relationship between viral respiratory diseases, environmental factors and development of bacterial and mycoplasmal pneumonia in dogs. In cats, bacterial pneumonia is less commonly identified than is inflammatory feline bronchial disease, though it

might be overlooked due to similarities in clinical presentation and diagnostic findings.

Classification of Bacterial Pneumonia

Aspiration

Aspiration pneumonia (AP) results from the inadvertent inhalation of gastric acid, oropharyngeal secretions, and/or ingesta and remains a common cause of bacterial pneumonia, accounting for roughly 23% of clinical diagnoses in a study of human patients admitted to the ICU¹. Though the true incidence of AP related to all causes is not well described in veterinary medicine, the incidence of post-anesthesia or sedation AP was reported to be 0.17% in one large multi-institution study.² Other than anesthesia, various conditions predispose to this disease. Risk factors that have been identified for the development of aspiration pneumonia include esophageal disease, refractory vomiting, seizures, and laryngeal dysfunction among others³ (Table 1).

In a healthy animal, physiologic and anatomic features reduce the chance of aspiration. During a normal swallow, fluid and food is propelled caudally in the oropharynx and through the upper esophageal sphincter by contraction of the oral cavity, pharynx and tongue. Concurrently, the epiglottis retracts to cover the laryngeal aditus and protect the trachea from particulate inhalation. Finally, adduction of the arytenoid cartilages contributes to further occlusion of the upper airways. Any process impeding these primary defenses or inhibiting normal swallowing reflexes greatly enhances the likelihood of aspiration.

Aspiration injury results from inhalation of either sterile, acidic gastric contents (resulting from vomiting or gastric regurgitation) or of septic material either from gastric or oral secretions. Irritation induced by acid inhalation promotes a local environment where bacterial colonization can develop and lead to bacterial pneumonia.⁴ The severity of disease varies depending on the quantity and nature of the material aspirated as well as the length of time between the event and diagnosis. Conscious animals with intact airway reflexes tend to cough and prevent massive aspiration injury. Animals under anesthesia or with reduced airway reflexes due to neurologic disorders are less likely to cough in response to the aspiration event and are, therefore, more likely to develop diffuse pulmonary infiltrates and serious lung injury. In many instances, aspiration injuries occur under general anesthesia and it should be noted that the presence of a cuffed endotracheal tube does not prevent inadvertent aspiration. Studies have demonstrated that concurrent use of cisapride with a proton-pump inhibitor reduces the incidence of gastroesophageal reflux under anesthesia^{5,6} and therefore might reduce the likelihood of aspiration pneumonia.

Canine Infectious Pneumonia

Infectious, or 'community acquired' pneumonias in dogs often begin with viral colonization and infection of the upper respiratory tract with canine respiratory coronavirus, adenovirus, herpesvirus, pneumovirus, parainfluenza virus or others⁷. Often, such diseases are acute and self-limiting, but in a subset of dogs, inflammation associated with these organisms immobilizes the host's immune defenses and predisposes to infection with other (often bacterial) respiratory pathogens⁸. Many bacteria have been implicated in canine infectious respiratory disease, though special focus has been directed towards *Streptococcus* (specifically *equi* subsp. *zooepidemicus* and *canis*), *Mycoplasma cynos*, and *Bordetella bronchiseptica*.⁹

Canine infectious respiratory disease (CIRD) is especially prevalent in dogs naïve to the pathogens and exposed in over-crowded, stressful environments such as animal shelters, boarding kennels, and treatment facilities, though it is important to remember that all dogs remain susceptible to these pathogens in any environment. The pathophysiology associated with this disease will be discussed later in this chapter (Box 1).

Foreign Body

Inhaled foreign bodies carry mixed bacterial and fungal organisms into the lung and are associated with focal or lobar pneumonias that are often initially responsive to antimicrobial medications but relapse shortly after discontinuation of therapy^{10,11}. Foreign bodies reported in the veterinary literature include grass awns, plant or plastic materials¹¹. Organisms associated with grass awn inhalation include *Pasteurella*, *Streptococcus*, *Nocardia*, *Actinomyces* and anaerobic bacteria, among others.¹⁰⁻¹² Most often foreign material remains at the carina or enters caudodorsal principle bronchi (accessory, right and left caudal lobar bronchi).

Features associated with pulmonary foreign bodies include:

- Young, sporting breeds
- Environmental exposure to grass awns
- Focal, recurrent radiographic alveolar pattern
- History of other cutaneous or visceral foreign bodies
- Spontaneous pneumothorax or pyothorax

Importantly, normal thoracic radiographs do not rule out the possibility of an airway foreign body and even computed tomography can fail to identify an affected bronchus.¹² Chronic pulmonary foreign bodies are associated with marked inflammation that can lead to massive airway remodeling and bronchiectasis, which, when seen on radiographs, should raise the degree of suspicion for foreign body.¹⁰

Nosocomial

Ventilator-associated pneumonia (VAP) is a common cause of hospital-acquired pneumonia in people though there are few reports in the veterinary literature. Colonization of the oropharynx by pathogenic and multidrug resistant bacteria occurs and the endotracheal tube acts as a conduit to transmit pathogens into the airways, which leads to tracheobronchitis and potentially pneumonia. In addition, any animal with a compromised respiratory tract or serious systemic disease is particularly prone to development of infectious airway disease while hospitalized.

The use of mechanical ventilation in human patients raises the risk of nosocomial infection by 6-20 fold.¹³ No published studies assess the risk in ventilated veterinary patients, though a study investigating difference in bacterial sensitivity between ventilated and non-ventilated animals suggested that dogs requiring mechanical ventilation were more likely to be infected with bacteria resistant to the antimicrobials most commonly used empirically to treat pneumonia in veterinary practice.¹⁴ This parallels the increase in incidence of multidrug resistant VAP in human medicine.¹³ In a recent outbreak of *Acinetobacter calcoaceticus*-*Acinetobacter baumannii* complex infections in a teaching hospital, nine of eleven animals were suspected of developing pneumonia due to contaminated equipment used during general anesthesia.¹⁵

Immune dysfunction

Both the innate and adaptive immune systems protect against the development of infectious airway disease, and a breakdown in either increases the likelihood of opportunistic infection. (Table 2) Congenital immunodeficiencies have been recognized that make an animal particularly sensitive to infectious organisms. Young animals are especially prone to the development of bacterial pneumonia due to their naïve immune system, and when coupled with alterations to the innate immune system such as primary ciliary dyskinesia (PCD), complement deficiency or bronchiectasis (congenital or acquired), the risk of life-threatening infection increases tremendously. See Veterinary Clinics of North America September 2007, Vol 37 (5): pp 845-860 for a comprehensive review of Respiratory Defenses in Health and Disease.

Any cause of systemic immunocompromise increases the risk for bacterial pneumonia, and any additional alterations to the body's natural defense mechanisms dramatically increase the risk. Specifically, medications such as chemotherapy, immunosuppressive therapy or antitussive therapy increase the likelihood of bacterial

pneumonia. Underlying respiratory viruses or systemic viruses such as FeLV and FIV have the potential to enhance the severity of respiratory illness.

Clinical Signs

Clinical signs of bacterial pneumonia vary depending on its etiology, severity and chronicity. They can be acute or peracute in onset or can display an insidious onset, resulting in chronic illness, particularly in animals with preexisting chronic airway disease. Early in disease, mild signs such as an intermittent, soft cough might be the only evidence of disease. As infection spreads, clinical signs worsen and often include a refractory, productive cough, exercise intolerance, anorexia and severe lethargy. Owners might note a change in the respiratory pattern, with increased panting or rapid breathing and in cases of severe infection, cyanosis and orthopnea can be observed. In general, these systemic signs are more often recognized in dogs than in cats.

Cats with pneumonia can display similar clinical signs to dogs, although the cough can be misinterpreted as a wretch or vomit by owners. Clinical signs and radiographic findings (such as right middle lobar consolidation or collapse) can also be considered suggestive of inflammatory airway disease rather than pneumonia.¹⁶ As disease worsens, cats can become tachypneic with short, shallow breaths and nasal flaring.¹⁷ Rarely do cat owners notice exercise intolerance associated with bacterial pneumonia.

Physical Examination

As with the history and clinical signs of bacterial pneumonia, physical examination findings vary greatly with the state and severity of disease. Dogs or cats with mild disease might have no abnormalities detected on physical exam. A change in the respiratory pattern, with an increase in rate and effort, can be an early clue to the diagnosis. The clinician must pay close attention to thoracic auscultation because adventitious lung sounds (crackles and wheezes) can be subtle, focal or intermittent. In many cases, only harsh or increased lung sounds are detected rather than crackles.¹⁸ Physical examination should assess for evidence of upper airway signs (e.g. nasal congestion or discharge) that can result from lower airway infection, either as an extension of epithelial infection or from nasopharyngeal regurgitation of lower airway secretions. Thorough auscultation of the trachea and upper airway is important for detecting upper airway obstructive disease that could predispose to pneumonia.

Animals with bacterial pneumonia generally present with mixed inspiratory and expiratory signs, similar to those seen with other

diseases of the pulmonary parenchyma. Fever is detected in 16-50% of cases, therefore it is not a reliable indicator of disease.^{8,16,18-20}

Diagnosis

Bacterial pneumonia implies sepsis of the lower airway and lungs; consequently the diagnosis is confirmed by demonstrating septic suppurative inflammation on airway cytology obtained through bronchoalveolar lavage (BAL) or tracheal wash, along with a positive microbiology culture. In some cases, this is completed easily and yields results consistent with clinical suspicion. Unfortunately, financial limitations or anesthetic concerns sometimes inhibit the ability to collect samples needed to confirm a bacterial infection, and in those cases a clinical diagnosis of bacterial pneumonia might be presumed based on available information.

A clinical diagnosis of bacterial pneumonia should be reached after obtaining compelling evidence to suggest a bacterial cause for the animal's clinical signs (after excluding other etiologies), and confirmed by resolution of signs following appropriate antimicrobial therapy. Acute bacterial pneumonia is a common diagnosis in the small animal clinic and can often be easily identified, however early and chronic pneumonias are more challenging to recognize because clinical signs can be subtle.

Hematology

The complete blood count is a useful diagnostic test in animals with respiratory signs. Bacterial pneumonias are often associated with an inflammatory leukogram, characterized primarily by a neutrophilia, with or without a left shift and variable evidence of toxic changes^{12,21}, although the absence of inflammatory change does not exclude the possibility of pneumonia.^{8,18} Furthermore, the leukogram and differential can provide clues that suggest bacterial pneumonia is less likely. For example, eosinophilia in an animal with respiratory signs would be more suggestive of eosinophilic bronchopneumopathy, granulomas or parasitic lung diseases as an underlying etiology than a bacterial cause. The erythrogram and platelet evaluation are generally not helpful in determining a bacterial etiology of respiratory disease.

A biochemistry panel and urinalysis do not always contribute to the diagnosis of bacterial pneumonia but can provide clues to the presence of metabolic or endocrine diseases that could make the development of bacterial pneumonia more likely. Similarly, fecal flotation, sedimentation, Baermann or heartworm test do not provide evidence for bacterial pneumonia but can be helpful in excluding parasitic pneumonia in areas where these organisms are endemic. Cats with

respiratory conditions should be screened for FeLV and FIV to detect systemic causes of immunosuppression.

Pulmonary Function Testing

Arterial blood gas analysis is a useful test to measure the lung's ability to oxygenate. Ideally, for animals with significant respiratory compromise, arterial blood samples should be collected and analyzed to determine the severity of pulmonary disease. Furthermore, trends in arterial oxygen partial pressure can be used to track progression or resolution of disease. In many cases, blood gas analysis is not available or patient factors preclude the acquisition of samples. Pulse oximetry is a quick, noninvasive evaluation of oxygen delivery to body tissues that measures percentage of hemoglobin saturation with oxygen. It provides only a crude assessment of oxygenation and is subject to variability, however trends in hemoglobin saturation can provide additional clinical support to progression or resolution of disease. Additionally, pulse oximetry provides a practical measure of oxygen desaturation during anesthesia for airway lavage and should be monitored closely during this procedure.

Thoracic Radiography

Thoracic radiographs are crucial diagnostic tests in the evaluation of lower airway and pulmonary parenchymal disease. Radiographic evidence of bacterial pneumonia can appear as a focal, multifocal or diffuse alveolar pattern, though early in the disease process infiltrates can be primarily interstitial.^{16,22} (Figures 1 and 2) Ventral lung lobes are most commonly affected in aspiration pneumonia, and a caudodorsal pattern would be expected with inhaled foreign bodies or hematogenous bacterial spread. A lobar sign is often seen in cases of aspiration pneumonia in which the right middle lung lobe is affected. (Table 3)

Three view thoracic radiographs (left lateral, right lateral and either dorsoventral or ventrodorsal views) should be obtained when screening for pneumonia because differential aeration associated with positional atelectasis can either mask or highlight pulmonary changes. For example, a radiograph taken in left lateral recumbency is preferred when aspiration is suspected because it will increase aeration of the right middle lung lobe, the most commonly affected lobe.

Diffuse radiographic involvement would be expected to suggest more severe disease, though radiographic changes lag behind clinical disease. Consequently bacterial pneumonia cannot be ruled out in animals with acute onset of clinical signs and unremarkable radiographs.¹²

Advanced Imaging

Advanced imaging is rarely necessary in the diagnosis of uncomplicated bacterial pneumonia, though it can be very helpful in more complicated cases. Thoracic ultrasound can be used to characterize peripheral areas of consolidation and to obtain fine-needle aspirates for cytology. Cytology is often helpful in distinguishing inflammation from neoplastic or fungal disease. Additionally, sonographic evaluation can be useful in the detection of superficial foxtail foreign bodies when they remain in the periphery of the lobe.¹² (Figures 3 and 4)

Computed tomography (CT) provides greater detail and resolution of lesions within the pulmonary parenchyma and gives the clinician better spatial information regarding the severity and extent of pulmonary involvement. In particular, CT is much better at identifying the presence and extent of bronchiectasis in comparison to thoracic radiography. In some cases, CT can be useful to identify migration tracts associated with inhaled foreign bodies.¹² Unfortunately, in most cases general anesthesia is required for CT acquisition and prolonged recumbency can lead to atelectasis, which is difficult to differentiate radiographically from infiltrates. Repeating the CT in a different position after providing several maximal inspirations can alleviate atelectasis. Nuclear scintigraphy can be useful for the evaluation of ciliary dyskinesia, though secondary causes of mucociliary stasis (i.e. infection with *Mycoplasma* or *Bordetella*, exposure to smoke) must be excluded before assuming the diagnosis of primary ciliary dyskinesia (PCD). Due to the time necessary for image acquisition, magnetic resonance imaging (MRI) is not commonly used for the diagnosis of most respiratory diseases. Positron emission tomography (PET) has not been evaluated for use in bacterial pneumonia, though it might be useful in evaluating patients with atypical infiltrates or mass lesions when a definitive diagnosis is not forthcoming.

Bronchoscopic evaluation

Examination of the trachea and bronchial tree should be performed systematically. The endoscopist should note the color and character of the mucosa and any airway sections, making sure to evaluate all branches of the lower airways for evidence of foreign bodies, bronchiectasis or collapse (diffuse or focal changes). Airway mucosa in a normal animal should be pale pink with visible mucosal and pulmonary vessels. Airway bifurcations should appear as narrow, crisp mucosal margins.

Animals with pneumonia can have hyperemia of the epithelium, prominent mucosal vessels and evidence of airway inflammation,

appearing as rounded, thickened airway bifurcations and airway nodules. Airway secretions are usually opaque, therefore viscous and discolored (brown, yellow-green, or red-tinged) secretions can be indicative of inflammation or pneumonia.

Airway sampling

When available, bronchoalveolar lavage (BAL) is preferred for collection of a lower airway sample over tracheal wash because the trachea and carina are not sterile, even in healthy dogs.²³ Additionally, the sensitivity for detecting cytologic features of sepsis is greater with BAL than tracheal wash.¹⁹ However, when only a tracheal wash specimen can be obtained, due to the lack of equipment for BAL or because of patient instability, collection of a lower airway sample is desirable to identify infecting bacteria and to determine appropriate antimicrobial therapy through susceptibility testing. Oropharyngeal swabs are not suitable substitutes for making a diagnosis of pneumonia.

BAL cell counts in animals with bacterial pneumonia are markedly higher than in dogs with chronic bronchitis or other respiratory disease.²¹ Septic, suppurative inflammation is a reliable indicator of bacterial pneumonia in dogs²¹ and is likely indicative of bacterial pneumonia in cats. In those cases that lack evidence of airway sepsis (intracellular bacteria), BAL cytology generally reveals suppurative or mixed inflammation.²⁰ It is important to note that animals with *Mycoplasma* pneumonia can have positive culture in the absence of cytologic evidence of sepsis.

In animals with suspected or confirmed foreign bodies, a BAL sample should always be obtained from the affected airway as well as an additional site, with both submitted individually for cytologic analysis. Airway bacteria are more likely to be seen in the cytologic sample from the site of the foreign body than from an alternate site.¹¹ Furthermore, cytology of BAL samples obtained from multiple lobes can reveal different findings, even in cases of sterile inflammatory diseases like feline bronchial disease, thus reliance on a single segment BAL cytology could lessen the chance of yielding diagnostic results.²⁴

Microbiology

Diagnosis of bacterial pneumonia relies on identification of septic inflammation in conjunction with a positive bacterial culture. Typically, aerobic and *Mycoplasma* culture and sensitivity are requested, and in cases with markedly purulent secretions or a history of known aspiration or foreign bodies, anaerobic cultures should also be requested. Samples should be refrigerated in sterile containers until submitted. If multiple alveolar segments are sampled during BAL,

these are usually pooled for culture submission. When anaerobic cultures are desired, BAL fluid should be inoculated into the appropriate transport media and kept at room temperature until submission.

Cultures should be performed whenever possible in order to guide appropriate antimicrobial therapy. With overly liberal use of antibiotics, increasing populations of resistant microbes are being identified – particularly in animals with hospital-acquired pneumonia.^{25,26} However, airway samples cannot be collected in all animals, and in those instances, recommendations regarding antimicrobial stewardship should be followed.²⁷

Bacteria commonly isolated from lung washes of cats or dogs with bacterial pneumonia include enteric organisms (*E. coli*, *Klebsiella* spp.), *Pasteurella* spp., coagulase positive *Staphylococcus* spp., beta-hemolytic *Streptococcus* spp., *Mycoplasma* spp. and *Bordetella bronchiseptica*.^{20,22} (Table 4)

Treatment

Treatment of bacterial pneumonia varies depending on the severity of disease, and appropriate antimicrobial therapy is essential. The International Society for Companion Animal Infectious Disease (ISCAID) has published guidelines for treatment of dogs and cats with respiratory infections and these should be consulted for further details about recommendations.²⁷ For stable animals with mild disease, outpatient therapy consisting of administration of a single, oral antibiotic is often all that is necessary. (Table 5) Ideally, antimicrobial choices should be based on culture and sensitivity results from airway lavage samples as resistance to antimicrobials selected empirically has been reported in up to 26% of cases.²⁸ For critically ill animals in which airways samples cannot be obtained, blood cultures might be considered, though there is a lack of data on sensitivity in veterinary patients. Regardless, in cases of severe pneumonia, initial empiric therapy should be instituted while awaiting culture results. Traditionally, antimicrobials have been administered for 3-6 weeks, and at least 1-2 weeks beyond the resolution of clinical and/or radiographs signs of disease, although there is no evidence to support this practice. ISCAID recommendations suggest that shorter duration might be appropriate, but there is little data to support this suggestion. One observational study found similar radiographic and clinical cures in dogs treated with a short course of antibiotic (less than 14 days) in comparison to those that received longer duration of treatment.²⁹ Regardless of the intended duration of therapy, re-evaluation within

10-14 days of starting treatment is important to determine response and to define optimal length of treatment.

Animals with more advanced disease require more intensive care, including hospitalization with intravenous fluids to maintain hydration. Adequate hydration is essential to facilitate clearance of respiratory exudates. Nebulization to create liquid particles that enter the lower airways (<5 microns in size) can also enhance clearance of secretions. Nebulizer types include ultrasonic devices, compressed air nebulizers, and a mesh nebulizer. Nebulization with sterile saline can be achieved by directing the hosing from the nebulizer into a cage or animal carrier covered in plastic. Depending on how viscous secretions are, therapy can be provided for 15-20 minutes 2-4 times daily. In many cases, nebulization coupled with coupage can help the animal expectorate airway secretions, although no specific studies in veterinary medicine have evaluated this technique. Coupage is performed by cupping the hands and gently, rhythmically pounding on both lateral thoracic walls in a dorsal to ventral and caudal to cranial direction. Coupage should not be performed in animals with regurgitation because any increase in intrathoracic pressure could exacerbate regurgitation and subsequent re-aspiration.

Supplemental oxygen is necessary for animals with moderate to marked hypoxemia (documented by a PaO_2 less than 80 mmHg or SpO_2 less than 94% on room air) in conjunction with increased respiratory effort. Oxygen supplementation at 40-60% is provided until respiratory difficulty lessens and the animal can be weaned to room air. Animals with refractory pneumonia that fail to improve on supplemental oxygen can succumb to ventilatory fatigue and need to be referred to an intensive care facility for mechanical ventilation.

Clinically, it seems that administration of an oral mucolytic agent such as N-acetylcysteine can be useful for animals with retention of thick respiratory secretions. In particular, this can be helpful in dogs with moderate to severe bronchiectasis that are prone to chronic or recurrent pneumonia. Decreasing the viscosity of airway secretions might improve expectoration of fluid and debris that accumulates in dependent airways, although no published information is available on use of mucolytics in animals. N-acetylcysteine is typically not used via nebulization due to risks of bronchoconstriction and epithelial toxicity. Under no circumstances is it appropriate to use cough suppressants (such as butorphanol or hydrocodone) in the management of bacterial pneumonia, particularly when it is complicated by bronchiectasis. By decreasing the cough reflex, these drugs perpetuate retention of mucus, debris and other material in the airways and therefore hinder clearance of infection. Also, furosemide should not be used because

drying of secretions will trap material in the lower airway and perpetuate infection.

In cases where aspiration pneumonia is suspected, strategies should be employed to reduce the chance of re-aspirating through appropriate treatment of the underlying condition. With disorders of esophageal motility, upright feedings of either slurry or meatballs can enhance esophageal transit. Furthermore, diets low in fat can increase gastric emptying. In patients with refractory vomiting, antiemetic and prokinetic agents can be employed to reduce the episodes of vomiting. Drugs such as maropitant (Cerenia®; 1 mg/kg IV or SQ once daily) or ondansetron (Zofran®; 0.3-1 mg/kg IV or SQ once to twice daily) act peripherally or centrally to decrease the urge to vomit and are safe to use in both cats and dogs.

The role of antacids in management of aspiration pneumonia remains controversial. On one hand, by neutralizing the pH of gastric secretions, animals with refractory vomiting or regurgitation are less likely to succumb to chemical injury related to aspiration. However, in cases treated with acid suppression, the aspirant could be more likely to contain a greater concentration of bacteria that can colonize the airways and lead to bacterial pneumonia. Though treatment with proton pump inhibitors has been shown to reduce the incidence of acid reflux events in both dogs and cats undergoing anesthesia^{5,30}, no controlled studies have assessed the severity of aspiration pneumonia or relative risk of using antacid therapy in dogs or cats as a preventative measure.

Because radiographic findings lag behind clinical disease, recheck radiographs are not helpful early in the disease process, though they are useful to document resolution of disease and should be obtained either before or within a week of discontinuation of antimicrobial therapy. In cases of refractory pneumonia, recheck radiographs midway through therapy can assess resolution or progression of disease and will help to guide further diagnostics and therapy.

Serum biomarkers such as acute phase proteins are associated with inflammatory disease. Being relatively nonspecific, these biomarkers are not clinically useful for diagnosis but might be helpful in determining treatment response, facilitating better antimicrobial stewardship by suggesting resolution of disease more rapidly than thoracic radiographs.³¹ Further studies are required to establish their utility in management of pneumonia.

In animals suspected of having contagious or multidrug resistant pathogens, appropriate contact precautions should be employed.

Isolation gowns, exam gloves and good hand washing technique along with appropriate quarantine facilities are essential to prevent transmission of disease to other animals or members of the healthcare team.

Prognosis

Prognosis for animals with bacterial pneumonia varies depending on the severity of disease, the animal's immunocompetence and the virulence of the infectious agent. In general, between 77-94% of patients diagnosed with pneumonia are discharged from the hospital.^{8,28,32} No large long-term studies assess the overall prognosis of animals with MDR bacteria or recurrent pneumonia. Presumably, the outcome associated with these cases will be worse. In a recent case series of presumed nosocomial multidrug resistant *Acinetobacter calcoaceticus*-*Acinetobacter baumannii* complex infections, 8/11 animals with pneumonia died or were euthanized as a consequence of their disease.¹⁵

CASE STUDIES

Case Study 1

A 7-year-old MC Bichon Frisé presented for a chronic cough.

History

Six-year history of progressive cough since adoption. The cough is described as non-productive, worse in the morning and exacerbated by aerosols and heavy fragrances. Previous treatment with theophylline and doxycycline have not lessened the severity of cough.

Physical examination

Temperature (101.9°F, 38.9°C), pulse (72), and respiratory rate (32) were normal. No heart murmur but soft crackles were ausculted on inspiration. A cough was elicited on tracheal palpation.

Diagnostic evaluation

Chronic cough in a small breed dog is often associated with airway collapse or chronic bronchitis, however infectious and neoplastic disease must remain on the differential list. Congestive heart failure is unlikely in this case given the lack of a heart murmur and normal heart rate.

A white blood cell count was normal (6650 cells/μl) with 4722 neutrophils. Thoracic radiographs revealed dynamic lower airway narrowing between lateral projections and a diffuse prominent bronchointerstitial pattern, most prominent in the caudal thorax. (Figure 5). The larynx appeared to have normal function at anesthetic induction. Bronchoscopy revealed mild to moderate dynamic lower airway collapse and bronchiectasis of caudodorsal bronchi along with airway exudate. Bronchoalveolar lavage samples were hypercellular on cytology (2500 cells/μl) and revealed septic suppurative inflammation (55%, normal 5-8%) with degenerate neutrophils. Bacterial cultures

were positive for *Pasteurella dagmatis* and *Fusobacterium* sp. In this case, chronic inflammatory airway disease likely contributed to the dog's bronchiectasis, which then predisposed to bronchopneumonia.

Case Study 2

A 5-year-old MC DMH was presented for evaluation of acute respiratory distress.

History

Lethargy and anorexia had been noted 3 days prior to the onset of respiratory signs.

Physical examination

Temperature (101.6°F, 38.7°C) and pulse (210) were normal. Tachypnea was noted (respiratory rate 60) along with increased respiratory effort on inspiration and expiration. Diffuse expiratory wheezes were ausculted.

Diagnostic evaluation

Acute onset of respiratory difficulty in a cat is most commonly related to inflammatory airway disease. The physical examination is consistent with this diagnosis, although it is uncommon for affected cats to demonstrate lethargy and anorexia. Infectious and neoplastic diseases were also on the differential diagnosis list, along with aspiration and foreign body pneumonia.

Thoracic radiographs demonstrated a focal opacity in the left caudal lung lobe and a diffuse bronchial pattern. (Figure 6) Complete blood count revealed a normal white blood cell count (8500 cells/μl) with a left shift (6800 neutrophils, 1000 bands). Bronchoscopy with lavage was performed. A moderate amount of airway hyperemia and edema was noted along with purulent material obstructing several airways. Bronchoalveolar lavage cytology had increased cellularity (1500 cells/μl, normal 500 cells/μl) with neutrophilic inflammation (84%, normal 5-8%). Neutrophils contained dark blue granular debris, suspicious for sepsis. Aerobic and anaerobic cultures were negative, but a pure culture of *Mycoplasma* was isolated on special media. A diagnosis of *Mycoplasma* bronchopneumonia was made.

References

1. Leroy O, Vandenbussche C, Coffinier C, et al. Community-acquired aspiration pneumonia in intensive care units. Epidemiological and prognosis data. *American journal of respiratory and critical care medicine*. 1997;156(6):1922-1929.
2. Ovbey DH, Wilson DV, Bednarski RM, et al. Prevalence and risk factors for canine post-anesthetic aspiration pneumonia (1999-2009): a multicenter study. *Vet Anaesth Analg*. 2014;41(2):127-136.
3. Tart KM, Babski DM, Lee JA. Potential risks, prognostic indicators, and diagnostic and treatment modalities affecting survival in dogs with presumptive aspiration pneumonia: 125 cases (2005-2008). *J Vet Emerg Crit Care (San Antonio)*. 2010;20(3):319-329.
4. Mitsushima H, Oishi K, Nagao T, et al. Acid aspiration induces bacterial pneumonia by enhanced bacterial adherence in mice. *Microbial Pathogenesis*. 2002;33(5):203-210.
5. Zacuto AC, Marks SL, Osborn J, et al. The influence of esomeprazole and cisapride on gastroesophageal reflux during anesthesia in dogs. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2012;26(3):518-525.
6. Ogden J, Ovbey D, Saile K. Effects of preoperative cisapride on postoperative aspiration pneumonia in dogs with laryngeal paralysis. *J Small Anim Pract*. 2019;60(3):183-190.
7. Brownlie J, Mitchell J, Walker CA, Erles K. Mycoplasmas and Novel Viral Pathogens in Canine Infectious Respiratory Disease. *J Vet Intern Med (Seattle)*. 2013.
8. Radhakrishnan A, Drobatz KJ, Culp WTN, King LG. Community-acquired infectious pneumonia in puppies: 65 cases (1993-2002). *Javma-J Am Vet Med A*. 2007;230(10):1493-1497.
9. Taha-Abdelaziz K, Bassel LL, Harness ML, Clark ME, Register KB, Caswell JL. Cilia-associated bacteria in fatal Bordetella bronchiseptica pneumonia of dogs and cats. *Journal of veterinary diagnostic investigation : official publication of the American Association of Veterinary Laboratory Diagnosticians, Inc*. 2016;28(4):369-376.
10. Workman HC, Bailiff NL, Jang SS, Zinkl JG. Capnocytophaga cynodegmi in a rottweiler dog with severe bronchitis and foreign-body pneumonia. *Journal of clinical microbiology*. 2008;46(12):4099-4103.
11. Tenwolde AC, Johnson LR, Hunt GB, Vernau W, Zwingerberger AL. The role of bronchoscopy in foreign body removal in dogs and cats: 37 cases (2000-2008). *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2010;24(5):1063-1068.

12. Schultz RM, Zwingenberger A. Radiographic, Computed Tomographic, and Ultrasonographic Findings with Migrating Intrathoracic Grass Awns in Dogs and Cats. *Veterinary Radiology & Ultrasound*. 2008;49(3):249-255.
13. Craven DE, Hjalmarson KI. Ventilator-associated tracheobronchitis and pneumonia: thinking outside the box. *Clin Infect Dis*. 2010;51 Suppl 1:S59-66.
14. Epstein SE, Mellema MS, Hopper K. Airway microbial culture and susceptibility patterns in dogs and cats with respiratory disease of varying severity. *J Vet Emerg Crit Care (San Antonio)*. 2010;20(6):587-594.
15. Kuzi S, Blum SE, Kahane N, et al. Multi-drug-resistant *Acinetobacter calcoaceticus*-*Acinetobacter baumannii* complex infection outbreak in dogs and cats in a veterinary hospital. *J Small Anim Pract*. 2016;57(11):617-625.
16. Levy N, Ballegeer E, Koenigshof A. Clinical and radiographic findings in cats with aspiration pneumonia: retrospective evaluation of 28 cases. *J Small Anim Pract*. 2019.
17. Egberink H, Addie D, Belak S, et al. *Bordetella bronchiseptica* infection in cats. ABCD guidelines on prevention and management. *Journal of feline medicine and surgery*. 2009;11(7):610-614.
18. Kogan DA, Johnson LR, Jandrey KE, Pollard RE. Clinical, clinicopathologic, and radiographic findings in dogs with aspiration pneumonia: 88 cases (2004-2006). *Journal of the American Veterinary Medical Association*. 2008;233(11):1742-1747.
19. Hawkins EC, DeNicola DB, Plier ML. Cytological analysis of bronchoalveolar lavage fluid in the diagnosis of spontaneous respiratory tract disease in dogs: a retrospective study. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 1995;9(6):386-392.
20. Johnson LR, Queen EV, Vernau W, Sykes JE, Byrne BA. Microbiologic and cytologic assessment of bronchoalveolar lavage fluid from dogs with lower respiratory tract infection: 105 cases (2001-2011). *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2013;27(2):259-267.
21. Peeters DE, McKiernan BC, Weisiger RM, Schaeffer DJ, Clercx C. Quantitative bacterial cultures and cytological examination of bronchoalveolar lavage specimens in dogs. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2000;14(5):534-541.
22. Cohn LA. Pulmonary Parenchymal Disease. In: Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine* :

- diseases of the dog and the cat*. Vol 2. 7 ed. St. Louis, Missouri: Saunders, Elsevier; 2010.
23. McKiernan BC, Smith AR, Kissil M. Bacterial isolates from the lower trachea of clinically healthy dogs. *Journal of the American Animal Hospital Association*. 1984;20:139-142.
 24. Ybarra WL, Johnson LR, Drazenovich TL, Johnson EG, Vernau W. Interpretation of multisegment bronchoalveolar lavage in cats (1/2001-1/2011). *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2012;26(6):1281-1287.
 25. Chalker VJ, Waller A, Webb K, et al. Genetic diversity of *Streptococcus equi* subsp. *zooepidemicus* and doxycycline resistance in kennelled dogs. *Journal of clinical microbiology*. 2012;50(6):2134-2136.
 26. Foley JE, Rand C, Bannasch MJ, Norris CR, Milan J. Molecular epidemiology of feline bordetellosis in two animal shelters in California, USA. *Preventive veterinary medicine*. 2002;54(2):141-156.
 27. Lappin MR, Blondeau J, Boothe D, et al. Antimicrobial use Guidelines for Treatment of Respiratory Tract Disease in Dogs and Cats: Antimicrobial Guidelines Working Group of the International Society for Companion Animal Infectious Diseases. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2017;31(2):279-294.
 28. Proulx A, Hume DZ, Drobatz KJ, Reineke EL. In vitro bacterial isolate susceptibility to empirically selected antimicrobials in 111 dogs with bacterial pneumonia. *J Vet Emerg Crit Care (San Antonio)*. 2014;24(2):194-200.
 29. Wayne A, Davis M, Sinnott VB, Bracker K. Outcomes in dogs with uncomplicated, presumptive bacterial pneumonia treated with short or long course antibiotics. *The Canadian veterinary journal / La revue veterinaire canadienne*. 2017;58(6):610-613.
 30. Garcia RS, Belafsky PC, Della Maggiore A, et al. Prevalence of Gastroesophageal Reflux in Cats During Anesthesia and Effect of Omeprazole on Gastric pH. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2017;31(3):734-742.
 31. Viitanen SJ, Lappalainen AK, Christensen MB, Sankari S, Rajamaki MM. The Utility of Acute-Phase Proteins in the Assessment of Treatment Response in Dogs With Bacterial Pneumonia. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2017;31(1):124-133.
 32. Kogan DA, Johnson LR, Sturges BK, Jandrey KE, Pollard RE. Etiology and clinical outcome in dogs with aspiration pneumonia: 88 cases (2004-2006). *Journal of the American Veterinary Medical Association*. 2008;233(11):1748-1755.

33. Viitanen SJ, Lappalainen AK, Koho NM, Pessa-Morikawa T, Ressel L, Rajamaki MM. Recurrent bacterial pneumonia in Irish Wolfhounds: Clinical findings and etiological studies. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2019;33(2):846-855.
34. McBrearty A, Ramsey I, Courcier E, Mellor D, Bell R. Clinical factors associated with death before discharge and overall survival time in dogs with generalized megaesophagus. *Journal of the American Veterinary Medical Association*. 2011;238(12):1622-1628.
35. Bedu AS, Labruyere JJ, Thibaud JL, et al. Age-related thoracic radiographic changes in golden and labrador retriever muscular dystrophy. *Veterinary radiology & ultrasound : the official journal of the American College of Veterinary Radiology and the International Veterinary Radiology Association*. 2012;53(5):492-500.
36. Watson PJ, Herrtage ME, Peacock MA, Sargan DR. Primary ciliary dyskinesia in Newfoundland dogs. *The Veterinary record*. 1999;144(26):718-725.
37. Watson PJ, Wotton P, Eastwood J, Swift ST, Jones B, Day MJ. Immunoglobulin deficiency in Cavalier King Charles Spaniels with Pneumocystis pneumonia. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*. 2006;20(3):523-527.
38. Jezyk PF, Felsburg PJ, Haskins ME, Patterson DF. X-linked severe combined immunodeficiency in the dog. *Clinical immunology and immunopathology*. 1989;52(2):173-189.
39. Foster SF, Martin P, Braddock JA, Malik R. A retrospective analysis of feline bronchoalveolar lavage cytology and microbiology (1995-2000). *Journal of feline medicine and surgery*. 2004;6(3):189-198.
40. Pesavento PA, Hurley KF, Bannasch MJ, Artiushin S, Timoney JF. A clonal outbreak of acute fatal hemorrhagic pneumonia in intensively housed (shelter) dogs caused by *Streptococcus equi* subsp. *zooepidemicus*. *Veterinary pathology*. 2008;45(1):51-53.
41. Mellema M. Viral Pneumonia. In: King LG, ed. *Textbook of Respiratory Disease in Dogs and Cats*. Vol 1. St. Louis, MI: Saunders; 2004:431-445.
42. Chalker VJ, Brooks HW, Brownlie J. The association of *Streptococcus equi* subsp. *zooepidemicus* with canine infectious respiratory disease. *Veterinary microbiology*. 2003;95(1-2):149-156.
43. An DJ, Jeoung HY, Jeong W, et al. A serological survey of canine respiratory coronavirus and canine influenza virus in Korean dogs. *The Journal of veterinary medical science / the Japanese Society of Veterinary Science*. 2010;72(9):1217-1219.

44. Knesl O, Allan FJ, Shields S. The seroprevalence of canine respiratory coronavirus and canine influenza virus in dogs in New Zealand. *New Zealand veterinary journal*. 2009;57(5):295-298.
45. Mitchell JA, Brooks HW, Szladovits B, et al. Tropism and pathological findings associated with canine respiratory coronavirus (CRCoV). *Veterinary microbiology*. 2013;162(2-4):582-594.
46. Kawakami K, Ogawa H, Maeda K, et al. Nosocomial outbreak of serious canine infectious tracheobronchitis (kennel cough) caused by canine herpesvirus infection. *Journal of clinical microbiology*. 2010;48(4):1176-1181.
47. Keil DJ, Fenwick B. Canine Respiratory Bordetellosis: Keeping up with an Evolving Pathogen. In: Carmichael LE, ed. *Recent Advances in Canine Infectious Disease*. International Veterinary Information Service; 2000.
48. Chalker VJ, Owen WM, Paterson C, et al. Mycoplasmas associated with canine infectious respiratory disease. *Microbiology*. 2004;150(Pt 10):3491-3497.
49. Priestnall S, Erles K. Streptococcus zooepidemicus: an emerging canine pathogen. *Veterinary journal*. 2011;188(2):142-148.
50. Foster SF, Martin P, Allan GS, Barrs VR, Malik R. Lower respiratory tract infections in cats: 21 cases (1995-2000). *Journal of feline medicine and surgery*. 2004;6(3):167-180.
51. Wood PR, Hill VL, Burks ML, et al. Mycoplasma pneumoniae in children with acute and refractory asthma. *Annals of allergy, asthma & immunology : official publication of the American College of Allergy, Asthma, & Immunology*. 2013;110(5):328-334 e321.

Figure legends

Figure 1. Dorsoventral (A) and right lateral (B) thoracic radiographs from a dog with an alveolar pattern in the cranioventral lung lobes, suggestive of aspiration. Note that in many cases the right middle lung lobe is most affected, which is best seen on a left lateral orthogonal view.

Figure 2. Dorsoventral (A) and right lateral (B) thoracic radiographs of a dog with a focal, patchy interstitial to alveolar pattern in the left cranial lung lobe. This dog was diagnosed with a foxtail foreign body, which was removed thoracoscopically via lung lobectomy.

Figure 3. A foxtail foreign body retrieved bronchoscopically from the left principle bronchus of a dog with chronic respiratory signs. Foxtails are endemic to the Western and Midwestern United States as well as some parts of Europe and are associated with mixed aerobic and

anaerobic infections. Fungal infections seem to occur as a consequence of bronchopulmonary foreign bodies less commonly.

Figure 4. CT image of a dog with severe, diffuse pneumonia resulting from a chronic foxtail foreign body (see figure 3). The foreign body was not visible on thoracic radiographs, though is clearly evident in the left principle bronchus on this image.

Figure 5. Thoracic radiographs revealing bronchiectasis and a diffuse prominent bronchointerstitial pattern, most prominent in the caudal thorax (Case study 1).

Figure 6. Thoracic radiographs revealing a focal opacity in the left caudal lung lobe and a diffuse bronchial pattern (Case study 2).

Table 1. Factors associated with aspiration pneumonia	
<ul style="list-style-type: none"> • Gastrointestinal disease <ul style="list-style-type: none"> o Refractory vomiting due to systemic or metabolic disease o Pancreatitis o Intussusception o Foreign body obstruction ● Ileus 	<ul style="list-style-type: none"> • Anesthesia <ul style="list-style-type: none"> o Prolonged anesthesia o Post procedural upper airway obstruction
<ul style="list-style-type: none"> • Esophageal disease <ul style="list-style-type: none"> o Megaesophagus o Esophageal motility disorder o Hiatal hernia o Esophageal stricture ● Esophagitis 	<ul style="list-style-type: none"> • Neurologic disease <ul style="list-style-type: none"> o Polyneuropathy o Myasthenia gravis o Seizure o Conditions leading to prolonged recumbency
<ul style="list-style-type: none"> • Cricopharyngeal dyssynchrony • Muscular dystrophy • Oropharyngeal dysphagia • Laryngeal disease • Tracheostomy 	<ul style="list-style-type: none"> • Breed <ul style="list-style-type: none"> o Brachycephalic breeds o Golden Retriever o Cocker Spaniel o English Springer Spaniel o Irish Wolfhound

Data from Refs 18,33-35

Table 2. Conditions leading to impaired immune function and resulting in increased risk of pneumonia		
	Congenital	Acquired
Innate	Primary ciliary dyskinesia Complement deficiency Leukocyte adhesion deficiency	Bronchiectasis Secondary ciliary dyskinesia
Adaptive	Immunoglobulin deficiency Severe combined immunodeficiency	Retrovirus infection (e.g. FIV, FeLV) Endocrine or metabolic disease (e.g. DM or HAC) Chemotherapy and other immunosuppressive

		therapy Splenectomy
--	--	------------------------

Data from Refs [36-38](#).

Table 3. Differential diagnoses for specific radiographic patterns

Lobar alveolar consolidation Aspiration pneumonia (cranioventral, right middle) Lung lobe torsion (cranial) Atelectasis secondary to mucus plugging (right middle most commonly)	Focal alveolar consolidation Airway foreign body Granuloma Primary pulmonary neoplasia (caudal lobes) Metastatic neoplasia Noncardiogenic pulmonary edema
Diffuse alveolar pattern Acute respiratory distress syndrome (ARDS) Congestive heart failure (perihilar in dogs) Fluid overload Eosinophilic bronchopneumopathy Coagulopathy Metastatic neoplasia Fungal pneumonia	Diffuse or focal interstitial pattern Early bacterial pneumonia Imminent congestive heart failure <i>Pneumocystis canis</i> infection Inhalant toxicity (e.g. Paraquat) Viral pneumonia

Table 4. Bacteria commonly isolated from airway samples of canine patients with pneumonia.

Organism	Percentage of isolates
<i>Bordetella bronchiseptica</i>	22-71%
<i>E. coli</i>	11-51%
<i>Klebsiella pneumoniae</i>	2-25%
<i>Pasteurella</i> spp.	3-21%
<i>Mycoplasma</i> spp.	30-70%
<i>Streptococcus</i> spp.	6-21%
<i>Staphylococcus</i> spp.	7-20%
<i>Anaerobes</i>	5-17%
<i>Enterococcus</i> spp.	4-11%

Data from Refs ^{8,20,28,39}

Table 5. Empiric antibiotic choice for patients with pneumonia	
Stable patient, mild clinical signs	<i>Monotherapy:</i> Doxycycline 5 mg/kg PO every 12 hours Amoxicillin-clavulanic acid 13.75mg/kg PO every 12 hours (dog) 62.5mg PO every 12 hours (cat)
Moderate clinical signs	<i>Monotherapy:</i> As above <i>Dual therapy:</i> Amoxicillin 22 mg/kg PO every 12 hours Ampicillin 22-30 mg/kg IV every 8 hours Clindamycin 10 mg/kg PO/SQ every 12 hours (dog) 10-15 mg/kg PO/SQ every 12 hours (cat) AND Enrofloxacin 10mg/kg PO/IV every 24 hours (dog) 5mg/kg PO/IV every 24 hours (cat) Pradofloxacin 7.5 mg/kg PO every 24 hours (cat) Amikacin 15mg/kg SQ every 24 h
Critical patient, severe clinical signs	<i>Dual therapy</i> As above <i>Monotherapy:</i> Piperacillin-tazobactam 50mg/kg IV every 6 hours Meropenem 24mg/kg IV every 24 hours Imipenem 10mg/kg IV every 8 hours

Data from Ref [27](#)

BOX 1: Canine Infectious Respiratory Disease (CIRD) Complex - changing the face of Kennel Cough

Canine infectious respiratory disease complex (formerly known as “kennel cough”) is a syndrome in which multiple pathogens, both viral and bacterial, coinfect either naïve, immunocompromised dogs or previously vaccinated dogs. This complex is multifactorial and it seems likely that both host and environmental factors play a role in the development of illness.⁴⁰ Organisms associated with this disease are ubiquitous, especially in overcrowded housing facilities such as animal shelters and training facilities. It is likely that stress induced by the new environment and exposure to novel pathogens both play a role in development of disease.

In most cases, respiratory signs are present for days to weeks and most animals show mild to moderate clinical signs. Typically viral infections cause either a bronchopneumonia or bronchointerstitial pneumonia due to their propensity to infect and damage type I pneumocytes.⁴¹ As the condition progresses, desquamation of the respiratory epithelium and aggregation of inflammatory cells further reduce the lungs’ natural defenses, increasing the potential for secondary bacterial colonization and infection.

Previous studies have implicated viral organisms such as canine adenovirus or canine parainfluenza⁴² as major participants in CIRD, although recent studies have proposed novel respiratory pathogens such as canine respiratory coronavirus^{7,43-45}, canine influenza virus⁴³, and canine herpesvirus⁴⁶ as additional important pathogens associated with CIRD. *Bordetella bronchiseptica*^{9,47}, *Streptococcus canis*, *Streptococcus equi* subsp. *zooepidemicus*⁴², and *Mycoplasma cynos*^{7,48} have been implicated as secondary bacterial infections associated with CIRD. *S. equi* subsp. *zooepidemicus* infections, in particular, have been associated with a rapidly progressive and often fatal hemorrhagic pneumonia.^{40,49} Of note, some strains identified in outbreaks of this pathogen have been identified as resistant to tetracycline antibiotics, often the drug of choice prescribed for other bacterial pathogens associated with this complex.

BOX 2: Feline Lower Respiratory Tract Infections

Organisms that have been reported as lower respiratory pathogens of cats include *Pasteurella* spp., *E. coli*, *Staphylococcus* spp., *Streptococcus* spp., *Pseudomonas* spp., *B. bronchiseptica* and *Mycoplasma* spp.⁵⁰, and specific attention has been paid to *Mycoplasma* spp. due to a possible association with the induction and exacerbation of asthma in adult and pediatric human patients⁵¹. However, the association between lower respiratory infection and chronic inflammatory lower airway disease in cats is unclear and is a topic of ongoing interest.

Mycoplasma species are considered normal flora in the upper respiratory tract and their role is controversial in lower respiratory tract infection. Because they are rarely identified cytologically and specific culture or PCR is needed to document the presence of these organisms, the role of *Mycoplasma* in cats (as well as in dogs) remains difficult to define.